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LETTER TO THE EDITOR

Viroj Wiwanitkit

Why is acute post-streptococcal glomerulonephritis more common in the pediatric population?

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To the editor

Acute glomerulonephritis is characterized by the presence of hematuria, proteinuria, and edema, and often by hypertension and acute renal failure.[1] Acute post-streptococcal glomerulonephritis (APSGN) is the prototypic disease of acute glomerulonephritis. It is seen after both streptococcal pharyngeal and skin infections, with latency periods from infection to presentation of 7–14 days and 14–21 days, respectively.[1] Approximately 90% of post-streptococcal glomerulonephritis occurs in young children.[1] Considerable knowledge has been accumulated regarding the characteristics of APSGN, and many attempts have been made to identify a streptococcal factor or factors responsible for triggering this disease.[2] However, the pathogenic mechanism behind APSGN remains largely unknown.[2] It is believed that APSGN is an immune-mediated disease, in which an immune complex containing a streptococcal antigen is deposited in affected glomeruli.[3] The role of humoral immunity is presumed to be mediated by the in-situ formation of nephritogenic streptococcal antigen-antibody complexes and circulating immune complexes,[4] whereas, in the cellular immune component, a role for delayed-type hypersensitivity has been suggested to contribute to the pathogenesis of APSGN.[4] Nephritis-associated plasmin receptor bound to the glomeruli may also contribute to the pathogenesis of APSGN via plasmin and complement activation.[5] Consideration of the nanostructure of the glomeruli in relation to the immune complexes could provide good information for a better understanding of the basic pathology of APSGN.

In the literature, some authors have reviewed the reported sizes of glomerular basement membrane (GBM) pores, as well as the molecular size of the streptococcus-Ig complex. The molecular size of the streptococcus-Ig complex is about 15 nm (10 nm for streptococcus group A[6] and 5 nm for immunoglobulin[7]). The GBM pore sizes in children and adults are 2.0–3.0 nm and 4.0–4.5 nm, respectively.[8] Therefore, the immune complex molecule can more easily be rodded into the glomerulus in children than in adults. The rodding in the children can be 1.3 (4.0/3.0) to 2.3 (4.5/2.0) times easier than in adults.

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V. Wiwanitkit (✉)
Department of Laboratory Medicine, Faculty of Medicine,
Chulalongkorn University, Bangkok 10330, Thailand
e-mail: wviroj@yahoo.com